

14 Radio & Lab.

CXR 525

Lateral CXR **532**

Urine report **534**

Stool examination **537**

Liver function test (LFTs) **538**

ALT **538**

AST **538**

Albumin **541**

Bilirubin **541**

Total protein **541**

CBC 544

RBC's **544**

HB **544**

MCH **545**

MCHC **545**

MCV **545**

HCT **546**

TLC **546**

Coagulation profile **548**

Platelets **548**

PT & INR **548**

PTT **548**

Alkaline phosphatase **549**

ESR **551**

ABG **553**

VISIT...

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Caliente.COM

قال تعالى:

قُلْ مَنْ يُحْكِمُ مِنْ ظُلُمَاتِ الْبَرِّ وَالْبَحْرِ
تَدْعُونَهُ تَضَرُّعًا وَخُفْيَةً
لَئِنْ أَنْجَانَا مِنْ هَذِهِ لَنَكُونَنَّ مِنَ الشَّاكِرِينَ
قُلِ اسْبِغْ بِمَحْنِكُمْ مِنْهَا
وَمِنْ كُلِّ كَرْبٍ ثُمَّ أَنْتُمْ مُتَشَرُّكُونَ

الأنعام ٦٣

524

Chest X-ray (CXR)

Principles:

1. Images are usually taken on **inspiration** with the patient standing in front of the cassette & the X-ray source behind (postero-anterior i.e. PA)
2. There are 4 densities : air, fat, water/soft tissue and bone

Air:

لم يعترض الأشعة فأحرقت الفيلم ← يظهر أسود

Others:

اعترضت الأشعة فلم تحرق الفيلم وظل أبيض كما هو ← تظهر أبيض



Normal chest x-ray

When interpreting a radiograph

- Use a systemic approach that works for you e.g. from outside to inside or inside-out
- But start by assessing the technical qualities of the image :
 1. **Rotation**: the sternal end of the clavicles should symmetrically overlie the transverse process of the 4th or 5th thoracic vertebra.
 2. **Inspiration**: there should 6 ribs visible anteriorly (or 10 posteriorly). Hyperinflation can be abnormal e.g. COPD.



Reference of this picture: A-Z chest radiology p 4

A well centred x-ray. Medial ends of clavicles are equidistant from the spinous process.

525

Radio & Lab. | Page 2

14

Two pictures following adequate respiratory efforts. The CXR now appears normal.

Reference of these pictures: A-Z chest radiology p 6

3. **Exposure** :

- an under-exposed image will be **too white**
الأشعة ضعيفة فاحرقش الفيلم كويس
- and over-exposed image will be **too black**
الأشعة شديدة فحرقت الفيلم أوي

both cause a loss of definition and quality

4. **Position**: the entire lung margins must be visible, especially the costophrenic angle.

Six complete anterior ribs (and ten posterior ribs) are clearly visible.

Reference of this picture: A-Z chest radiology p 5

526

Radio & Lab. | Page 3

14

The effect of varied exposure on the quality of the final image.

■ **Review of important anatomy**

A. Heart and mediastinum

1. **Assessment of heart size**

- The cardiothoracic ratio should be less than 0.5, i.e. A/B < 0.5.
- 1/3 should lie to the Rt. of the vertebral column, 2/3 to the left
- A cardiothoracic ratio of greater than 0.5 (in a good quality film) suggests cardiomegaly.
- It may appear **elongated** if the chest is hyper-inflated (COPD) or **enlarged** if the image is AP, HF, pericardial effusion

Reference of these pictures: A-Z chest radiology p 7

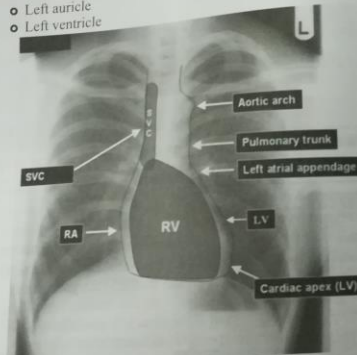
527



2. There are 4 mogules normally visible on the left border of the mediastinum that may help identify pathology if abnormal.

From superior to inferior:

- Aortic knuckle (aortic arch)
- Pulmonary outflow tract
- Left auricle
- Left ventricle



Reference of these pictures: A-Z chest radiology p 8

B. Trachea

- Should be **central or just to the Rt.**
- **Deviated** by collapse (towards the side of the lesion), by **tension** (away from the side of the lesion) or by **rotation** of the film
- The right wall of the trachea should be clearly seen as the so called right

528

• Para-tracheal stripe.

- The para-tracheal stripe is visible by virtue of the silhouette sign: air within the tracheal lumen and adjacent right lung apex outline the soft-tissue-density tracheal wall.
- Loss or thickening of the para-tracheal stripe intimates adjacent pathology.
- The trachea is shown in its normal position, just to the right of centre. The right para-tracheal stripe is clearly seen.



C. Hilum:

- The left hilum is higher than the Rt. Hilum (up to 1 cm), but they should be the same size and density.
- May be pulled down or up by fibrosis or collapse

• Enlarged hila:

- Nodes
- Pulmonary arterial HTN (± enlarged 2nd mogule)
- Bronchogenic carcinoma
- Calcification
- Previous T.B silicosis

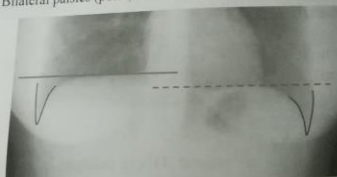
D. The diaphragm:

- The Rt. side is usually **slightly higher** (usually 1-1.5 cm)
- Each costo-phrenic angle should be sharply outlined. The outlines of both
- Both costophrenic angles are sharply outlined.
- The outlines of both hemidiaphragms should be clearly visible.

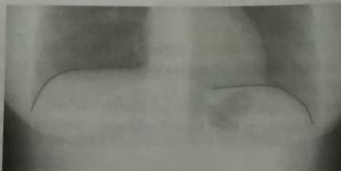
529



- Hemidiaphragms should be sharp and clearly visible along their entire length (except the medial most aspect of the left hemidiaphragm).
- The 'curvature' of both hemidiaphragms should be assessed to identify diaphragmatic flattening
- The highest point of a hemidiaphragm should be at least 1.5 cm above a line drawn from the cardiophrenic to the costophrenic angle.
- **Causes of a raised hemi-diaphragm:**
 - Lung volume loss.
 - Stroke.
 - Phrenic nerve palsy.
 - Hepatomegaly.
 - Sub-phrenic abscess.
 - Sub-pulmonic effusion.
- Bilateral palsies (polio, muscular dystrophy) cause hypoxia



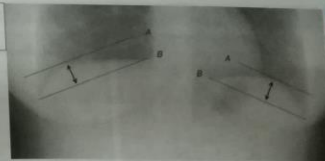
The right hemidiaphragm is 'higher' than the left. Both costophrenic angles are sharply outlined.



The outlines of both hemidiaphragms should be clearly visible.

530

Reference of the upper pictures: A-Z chest radiology p 8



Assess for diaphragmatic flattening. The distance between A and B should be at least 1.5 cm.

E. Lungs:

- Shadowing is described as **nodular**, **reticular** (network of fine lines شبكية) or **alveolar** (fluffy الغليظ)
- **Nodules:**
 1. **Neoplasia:** lung carcinoma, adenoma, metastases : often missed if small
 2. **Infections:** varicella pneumonia, hydatid, septic emboli
 3. **Granulomas:** miliary T.B, sarcoidosis, histoplasmosis
 4. **Pneumoconiosis** (except asbestosis).
- **Reticular shadows:**

Usually acute interstitial changes cardiac or non cardiac pulmonary interstitial oedema, atypical pneumonia or:

 - Fibrosis, TB, histoplasmosis.
 - Sarcoidosis, silicosis, asbestosis.
 - Fibrosing alveolitis, rheumatoid.
 - Wegner's.
 - SLE.
- **Alveolar shadows:**

Usually **pulmonary oedema** from LVF. Also:

 - Pneumonia
 - Renal or LF
 - Hge
 - ARDS, DIC
 - Drugs (heroin, cytotoxics)
 - Smoke inhalation
 - O₂ toxicity
 - Fat emboli ~ √days post-fracture
 - Head injury
 - Near drowning
 - Heat stroke

531

14

- **Ring shadows:**
 - Bronchiectasis
 - Cavitating lesion e.g. abscess (bacterial, fungal, amoebic)
 - Tm
 - Pulmonary infarct

- **White out of an entire hemithorax:**
 - Pneumonia
 - Massive pleural effusion
 - ARDS
 - After pneumonectomy

- **Air outside the lungs:** check for
 - Pneumothorax
 - Surgical emphysema (trauma, iatrogenic)
 - Gas under the diaphragm (perforated viscous, trauma, surgery)

F. Bones:

- Check the **clavicles** for bone density (loss of cortex in osteoporosis, or fracture)
- Check the **ribs** for fractures, **notching**, **absence** (surgery) and lesions (**metastases**).
- Check the vertebral column for **collapse** or **destruction**
- Check the shoulders for **fracture** and **arthritis**

• An apparently normal CXR?

- Check for **tracheal compression**, **absent breast shadows** (mastectomy), **double left heart border** (left lower lobe **collapse**, **fluid level** behind the ht. (HH, achalasia) and **paravertebral abscess**.

532

• Lateral CXR

14



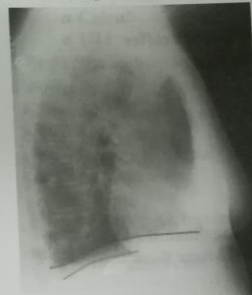
Key points

Central hilar structures

- Primary trunk
- Right upper lobe bronchus
- Left upper lobe bronchus
- Left pulmonary artery
- Aortic arch

Diaphragms

- The right hemidiaphragm is usually 'higher' than the left. The outline of
- the right can be seen extending from the posterior to anterior chest wall.
- The outline of the left hemidiaphragm stops at the posterior heart border.
- Air in the gastric fundus is seen below the left hemidiaphragm.



Source: A-Z chest radiology p 17

533

14

Laboratory investigations

1. Urine report

1. Volume.
2. Colour & aspect
3. Sp. gravity
4. Sugar
5. Albumin
6. Red cells
7. Pus cells
8. Casts
9. Organic salts
10. Bilirubin
11. Urobilinogen

1. Volume:

• Normally:

In hot climate: 600-900ml/24hr
In winter: may exceed 1 litre

• Polyuria

Causes:

- Excessive intake of fluids, tea & coffee.
- Diuretics.
- DM.
- Diabetes insipidus.
- Chronic renal failure.
- Hypercalcemia.
- Hysterical polydipsia.

• Oliguria

Causes:

- Severe hypotension causing diminished glomerular filtration from: e.g., shock, severe fluid loss from diarrhea.

534

14

- Damage of glomeruli or tubules.
- Acute glomerulonephritis
- Tubular necrosis
- Bilateral obstruction of ureters.

2. Protein

- Normal protein excretion is < 150mg/day, consisting of < 30mg/d of albumin.

• Causes of proteinuria

1. Orthostatic (postural): on upright position.

2. Functional

- Fevers
- Excessive
- Heat stroke or severe cold atmosphere
- Congestive heart failure

3. Kidney

- Nephrotic syndrome more than 3 g/24hrs
- Pyelonephritis
- Acute & chronic glomerulonephritis
- Tumors
- Renal T.B

4. Urinary tract

- Calculi
- UTI

3. Red cells: see haematuria in urology chapter

4. Pus cells

- Normally: should not exceed 3-6 cells/high power field.

• Causes:

- Infection (bacterial)
- Sterile pyuria:
 - T.B
 - During Ab treatment
 - Stones
 - Interstitial cystitis

535

14 5. Casts:

1. **Red cell casts:**
 - Think of glomerular bleeding e.g. acute glomerulonephritis.
2. **White cell casts:**
 - Seen in acute pyelonephritis
3. **Granular casts:**
 - Common in any renal disease but especially in
 - Acute tubular necrosis.
 - Acute glomerulonephritis.
4. **Hyaline casts:**
 - Seen in:
 - Normal urine, particularly if concentrated.
 - All forms of renal disease.
 - Fever.
 - Use of loop diuretics.
5. **Epithelial cell casts:**
 - Seen in:
 - Acute tubular necrosis.
 - Severe acute glomerulonephritis.

Urine Reports

	1	2	3	4
Volume	800-1200	2500	3500	---
Color & aspect	Amber yellow	Pale yellow	Watery	Red
Sp. Gravity	10.16-10.25	10.45	10.10	10.20
Red cells	Nil	Nil	+	+++
Pus cells	0-3	3	3	3
Casts	Nil	Lipoid ++	Gr. Epith	---
Organic salts	Very few or nil	Nil	Nil	Oxalates
Bilirubin	Nil	Nil	Nil	---
Urobilinogen	Trace	Nil	Nil	---
Diagnosis	Normal	DM + Nephrotic Syndrome	Ch.RF Ch.GN	Haematuria d.t. Oxaluria

2. Faeces:

1. Volume
2. Undigested food
3. Odour
4. Red cells
5. Consistency
6. Pus cells
7. Mucus
8. Fat content
9. Reaction
10. Unsplit fat
11. Remarks

1. Color:

- The normal colour varies from light brown to dark brown depending on the diet.
- Black stool** may be d.t.:
 - Upper GI bleeding** (Melena) + shiny
 - Iron:** black but **not shiny**
- Red blood:**
 - Lower GI bleeding** e.g. ulcerative colitis
 - From anal canal: **hemorrhoids**
- Dark green:**
 - Seen in **diarrhea**
- Gravish white:**
 - In **obstructive jaundice**
- Clay stools:**
 - Produced by excessive amounts of fat (**steatorrhea**)

2. Mucus:

- Excessive mucus is seen in:
 - Irritable colon**
 - Purgatives & Ab**
 - Neoplasms**
 - Inflammatory conditions**

14 3. Parasites:

- Ascaris, Ankylostoma, worms and segments of tenia, may be discovered on routine examination.

■ Reports on Faeces

	Normal	Amoebic dysentery	Malabsorption
Volume	Normal	Normal	Large
Odour	Normal	Offensive	Offensive
Consistency	Soft	Base shiny	Loose
Reaction	Alkaline	Acidic	Alkaline
Mucus	-	Excessive	+
Red cells	-	+++	-
Pus cells	-	++	Excessive
Fat content	-	-	+++
Undigested food	-	++	90%
Unsplit fat	+	-	-
Remarks		Active amoeba	

3. Liver function tests

■ Report

Total Bilirubin : N= 0.5-0.9mg %
 Total protein : N= 6.5-7.5mg %
 Globulin : N= 1.5-4g
 Alkaline phosphatase : N= 5-11 K.A unit
 S.G.P.T : N= 0-30
 S.G.O.T : N=
 Lactic Dehydrogenase : N= 200-700μ
 Albumin : N= 4-5.5g
 Prothrombin time : N= 10-15sec

1. ALT (SGPT)

■ Use

- Differential diagnosis of diseases of hepatobiliary system and pancreas
- Repeat testing to establish chronicity of viral hepatitis

- Generally parallels but lower than AST in alcohol-related diseases

■ Increased In

- See serum AST
- Obesity (not AST; modest increase to 1-3× ULN)
- Severe preeclampsia (both)
- Rapidly progressing acute lymphoblastic leukemia (both)
- Levels in females ~75% of those in males

■ Decreased In

- GU tract infection
- Malignancy
- Pyridoxal phosphate deficiency states (e.g., malnutrition, pregnancy, alcoholic liver disease)
- Others e.g. congenital deficiency

2. AST (SGOT)

■ Use

- Differential diagnosis of diseases of hepatobiliary system and pancreas

■ Interferences

- Increase due to hemolysis, lipemia
- Increase due to calcium dust in air (e.g., due to construction in laboratory)
- Increase due to enzyme activation during test
- Therapy with oxacillin, ampicillin, opiates, erythromycin
- Diabetic ketoacidosis**
- Beriberi
- Severe liver disease
- Chronic hemodialysis (reason unknown)
- Uremia—proportional to BUN level (reason unknown)

■ Increased In

■ Liver diseases:

- Acute viral **hepatitis** shows greatest increases; may be 20× to 100×.
- Rapid rise and decline suggests extrahepatic biliary disease.
- Congestion**, e.g., heart failure, cirrhosis, biliary obstruction, primary or metastatic cancer, granulomas, hepatic ischemia.

- Eclampsia.
- Hepatotoxic drugs and chemicals (e.g., carbon tetrachloride).
- Musculoskeletal disorders, including trauma, surgery, and IM injections
- Myoglobinuria
- Acute myocardial infarction
- **Others**
 - * Acute pancreatitis
 - * Intestinal injury (e.g., surgery, infarction)
 - * Local irradiation injury
 - * Pulmonary infarction (relatively slight increase)
 - * Cerebral infarction (increased in following week in 50% of patients)
 - * Cerebral neoplasms (occasionally)
 - * Renal infarction (occasionally)
 - * Drugs (e.g., heparin therapy, salicylates, opiates, tetracycline, chlorpromazine, isoniazid)
 - * Burns
 - * Heat exhaustion
 - * Mushroom poisoning
 - * Lead poisoning (not useful for screening)
 - * Hemolytic anemia
- **Marked Increase (>3000 U/L) In**
 - * Acute hypotension (e.g., AMI, sepsis, post-cardiac surgery)
 - * Toxic liver injury (e.g., drugs)
 - * Viral hepatitis
 - * Liver trauma
 - * Liver metastases
 - * Rhabdomyolysis
- **Decreased In**
 - Azotemia
 - Chronic renal dialysis
 - Pyridoxal phosphate deficiency states (e.g., malnutrition, pregnancy, alcoholic liver disease)
- **Normal In**
 - Angina pectoris
 - Coronary insufficiency

- Pericarditis
- Congestive heart failure without liver damage
- Varies <10 U/day in the same person.

3. AST/ALT (SGOT/SGPT) RATIO

(Normal = 0.7–1.4 depending on methodology.)

- **Use**
 - Differential diagnosis of diseases of hepatobiliary system and pancreas
- **Increased In**
 - Drug hepatotoxicity (>2.0)
 - Alcoholic hepatitis (>2.0 is highly suggestive; may be 6.0)
 - Cirrhosis (1.4–2.0)
 - Intrahepatic cholestasis (>1.5)
 - Hepatocellular carcinoma
 - Chronic hepatitis (slightly increased; 1.3)
- **Decreased In**
 - Acute hepatitis due to virus, drugs, toxins (with AST increased 3–10× upper limit of normal) (usually 0.65; ratio 0.3–0.6 is said to be a good prognostic sign but higher ratio of 1.2–1.6 is a poor prognostic sign)
 - Extrahepatic cholestasis (normal or slightly decreased; 0.8)1

4. Albumin

(Generally parallels total protein except when total protein changes are due to gamma globulins)

- **Use**
 - Marker of disorders of protein metabolism (e.g., nutritional, decreased synthesis, increased loss)
- **Increased In**
 - Dehydration (relative increase)
 - Intravenous (IV) albumin infusions
- **Decreased In**
 - Inadequate intake (e.g., malnutrition)
 - Decreased absorption (e.g., malabsorption syndromes)
 - Increased need (e.g., hyperthyroidism, pregnancy)
 - Impaired synthesis (e.g., liver diseases, chronic infection, hereditary analbuminemia)
 - Increased breakdown (e.g., neoplasms, infection, trauma)
 - Increased loss (e.g., edema, ascites, burns, hemorrhage, nephrotic syndrome, protein-losing enteropathy)

- Dilutional states (e.g., IV fluids, SIADH, psychogenic diabetes/water intoxication)

4. BILIRUBIN

- **Use**
 - Differential diagnosis of diseases of hepatobiliary system and pancreas and other causes of jaundice.
 - Jaundice becomes apparent clinically at 2.5 mg/dL.
- **Increased Direct (Conjugated) Bilirubin In**
 - Hepatic cellular damage. Increased conjugated bilirubin may be associated with normal total bilirubin in up to one-third of patients with liver diseases.
 - * Viral
 - * Toxic
 - * Alcohol related
 - * Drug related
 - Other effects (e.g., toxic, cholestasis)
 - Biliary duct obstruction
 - * Extrahepatic
 - * Intrahepatic
 - * Infiltrations, space-occupying lesions, e.g., Metastatic tumor, Abscess
 - Direct bilirubin
 - * 20–40% of total: more suggestive of hepatic than post-hepatic jaundice
- **Increased Unconjugated (Indirect) Bilirubin (Conjugated <20% of Total)**
 - Increased bilirubin production
 - * Hemolytic diseases (e.g., hemoglobinopathies, RBC enzyme deficiencies, disseminated intravascular coagulation [DIC], autoimmune hemolysis)
 - * Ineffective erythropoiesis (e.g., PA)
 - * Blood transfusions
 - * Hematomas
 - Hereditary disorders (e.g., Gilbert's disease, Crigler-Najjar syndrome)
 - Drugs (e.g., causing hemolysis)
- **Decreased In**
 - Drugs (e.g., barbiturates)

- **Interferences**
 - Presence of hemoglobin
 - Exposure to sunlight or fluorescent light
- **Decreased In**
 - Ingestion of certain drugs (e.g., barbiturates)
 - * 40–60% of total: occurs in either hepatic or post-hepatic jaundice
 - * >50% of total: more suggestive of post-hepatic than hepatic jaundice
 - Total serum bilirubin >40 mg/dL indicates hepatocellular rather than extrahepatic obstruction.

5. TOTAL PROTEIN

- **Use**
 - Screening for nutritional deficiencies and gammopathies
- **Increased In**
 - Hypergammaglobulinemias (mono- or polyclonal)
 - Hypovolemic states
- **Decreased In**
 - **Nutritional deficiency**, e.g.
 - * Malabsorption
 - * Kwashiorkor
 - * Marasmus
 - **Decreased or ineffective protein synthesis**, e.g.
 - * Severe liver disease
 - * Agammaglobulinemia
 - **Increased loss**
 - * Renal (e.g., nephrotic syndrome)
 - * GI disease (e.g., protein-losing enteropathies, surgical resection)
 - * Severe skin disease (e.g., burns, pemphigus vulgaris, eczema)
 - * Blood loss, plasmapheresis
 - **Increased catabolism**, e.g.
 - * Fever
 - * Inflammation
 - * Hyperthyroidism
 - * Malignancy
 - * Chronic diseases
 - **Dilutional**, e.g.
 - * IV fluid administration
 - * SIADH
 - * Water intoxication

14 Reports on liver functions

Total Bilirubin	8	3.5	1.7
SGPT	35	160	70
Albumin	4	4	1.4
Globulin	3.2	2.2	4.2
Total protein	2.4	6.2	5.6
Al. phosphatase	42	25	18
Lactic dehydrogenase	200	1200	600
P.T	35	30	40
P.concent. %	40%	60%	40%
Alpha-fetoprotein	-	-	-
Result	Obstructive jaundice	Infective hepatitis	Liver cirrhosis

4. Complete blood count (CBC)

1. Red blood cells

- RBC count is interpreted in conjugation with red cell indices, Hb & Hct
- Increased in:**
 - Severe dehydration
 - Certain myeloproliferative neoplasms e.g polycythemia vera
- Decreased in:** various types of anemia

2. Hemoglobin

- Normal range = 12-16 g/dl in women & 14-18 g/dl in men
- Decreased in all anemias, in most cases as consequences of another underlying disease or a deficiency (iron, folate, vit B12)
- Increased in:**
 - Physiologic response to high altitude due to O₂ tension
 - In advanced cardiac or respiratory disease
 - Certain myeloproliferative neoplasms, especially polycythemia vera

3. Mean corpuscular hemoglobin (MCH)

- MCH is the Hb concentration per RBCs count
- It has limited value in classifying anemias
- Normal range: 27-34 pg/red cells
- Increased in:**
 - Macrocytic anemias and infants as well as newborns
- Decreased in:**
 - Microcytic and normocytic anemias

References: Wallach 9th p 254

4. Mean corpuscular hemoglobin concentration (MCHC)

- MCHC is the Hct divided Hb
- Normal range: 31.5-36 g
- Decreased in:**
 - Macrocytic anemias & hereditary spherocytosis
 - Infants and newborns

References: Wallach 9th p 254

5. Mean corpuscular volume (MCV)

- MCV represents the average measurement of RBC volume
حجم خلية واحدة فقط
- Equals Hct/RBCs count
تساوي الحجم الكلي للكرات الحمراء على عددها
- Increased in:**
 - Macrocytic anemias
 - Liver disease
 - Alcoholism
 - Myelodysplastic syndromes
 - Hypothyroidism
 - Hemodialysis with high reticulocyte count
- Decreased in:**
 - Iron deficiency
 - Thalassemias
 - Anemia of chronic disease (chronic renal failure, rheumatic disease, infections, cancer, aplastic anemia)

- Lead poisoning
- Limitations
- NCV may artificially increase with marked leukocytosis, numerous large platelets. Marked hyperglycemia & marked leukocytosis
- MCV may be falsely decreased within vitro hemolysis of fragmentation of RBCs

Source: Wallach 9th p 245,255

6. Hematocrit (Hct)

- Hct is the ratio of spun RBCs to plasma, reflecting the volume of packed RBCs
- Hct = MCV x RBCs count
- Normal range: 37-47 % for women and 42-52 % for men

7. Total leukocytic count:

- Leukocytosis = TLC > 10,300/ μ L
- Leukopenia = TLC < 4,300/ μ L

A. Neutrophilia

- Means absolute neutrophil count > 7,500/ μ L (or >72%)

Primary neutrophilia

- Mean proliferative neoplasms
- Neutrophilic leukemia others

Secondary neutrophilia

- Acute infections
- Localized** (e.g. pneumonia, meningitis, tonsillitis, abscess, acute otitis media in children)
- Systemic** (e.g. septicemia)
- Inflammation, especially during diseases flare up
- Vasculitis, acute rheumatic fever, RA,
- Crohn's disease ulcerative colitis
- Chronic hepatitis
- In toxic conditions
- Metabolic (uremia, acidosis, Eclampsia, acute gout)
- Acute hge
- Acute hemolysis
- Tissue or tumor necrosis
- Acute MI
- Burns
- Gangrene
- Emotional stress & increased exercise
- Labor
- Smoking

B. Neutropenia

- <43% of leukocytes or an absolute neutrophil and band count <1600/ μ L

Causes:

- Decreased BM production (e.g. chemotherapy, acute leukemia, radiation therapy or accident, folic or vitamin B12 deficiency, etc)
- Increased production but decreased survival neutrophils
- Autoimmune neutropenia
- SLE and RA, others
- Viral infections:**
 - Infectious mononucleosis
 - HIV infections
 - Hepatitis
 - Influenza
 - Measles & Rubella
 - Psittacosis

6. Bacterial infections

- Sepsis
- Military T.B
- Typhoid & paratyphoid
- Brucellosis
- Rickettsial infections

7. Others e.g. malaria, Kala-azar

8. Drugs e.g. vancomycin, cephalosporins, Macrolides, Sulfa, antifungals, ACEIs, digoxin, diuretics, etc.)

C. Eosinophilia

- Def: count of >600/ μ L or > 8% of the differential count
- Primary eosinophilia**
 - Hyper-eosinophilic syndrome
 - Neoplastic disorders as chronic eosinophilic leukemia
- Secondary eosinophilia**
 - Allergic diseases
 - Parasitic infections, some fungal infections

Radio & Lab. | Page 24

- Collagen vascular disorders
- T-cell lymphomas, Hodgkin lymphoma
- Hypersensitivity
- Pneumonitis
- Adrenal insufficiency
- Immunologic reactions, transplant rejection
- Cholesterol embolism syndrome

5. Coagulation profile

1. Platelets

- **Normal range:** 140-440 (x10⁶ cells)
- **Increased in**
 - After acute hge, in malignancies, after splenectomy, severe trauma, infections, chronic inflammatory disorders during reactions
 - Myeloproliferative neoplasms
- **Decreased in**
 - Immune destruction such as: in ITP, reaction to certain drugs, aplastic anemia, leukemias, hypersplenism, DIC, etc.
 - Following chemotherapy, post-transfusion thrombocytopenia (develops after 5-10 days)

2. PT and INR

- **PT:** 9.6-12.4 seconds
- **INR = 1**
- **Use:**
 - evaluation of abnormalities of extrinsic coagulation mechanism (factor VII) & the common pathway (Factors II, V, X & fibrinogen)
 - PT is not sensitive in abnormalities of intrinsic pathway (factors XII, XI, IX & VII)
 - Evaluation of liver function reflecting abnormalities in factors VII, II, X, V
 - Factor V is not affected by oral anticoagulants
 - INR is used to monitor oral anticoagulants

548

- Radio & Lab. | Page 25
- **Interpretations**
 - Marked prolongation in liver → advanced dses
 - INR on oral anticoagulants (e.g. marivan) should be adjusted to 2-3
 - Combined abnormal PT and PTT is found in:
 - Medical oral anticoagulants, DIC, liver diseases, vit K deficiency. Massive transfusions.
 - Coagulation factor abnormalities

3. PTT

- Used in monitoring of therapy with unfractionated heparin
- Normal range: (22-36 seconds)
- Not used in monitoring of therapy with LMWH
- **Increased (>36 seconds) in:**
 - Therapy with unfractionated heparin
 - Single clotting factors
 - Inhibitors
- **Normal in:**
 - Thrombocytopenia and thrombocytopathies with associated clotting defect

Reference: Wallach 9th p 281,282

6. ALKALINE PHOSPHATASE (ALP)

- **Use**
 - Diagnosis of causes and monitoring of course of cholestasis (e.g., neoplasm, drugs)
 - Diagnosis of various bone disorders (e.g., Paget's disease, osteogenic sarcoma)
- **Interferences**
 - Intravenous injection of albumin; sometimes marked increase (e.g., 10× normal level) lasting for several days (placental origin); total parenteral nutrition (TPN)
 - Increased (30%) by standing at room or refrigerator temperature

549

Radio & Lab. | Page 26

- **Increased In**
 - **Bone origin** - increased deposition of calcium
 - **Liver disease** - any obstruction of biliary system
 - Nodules in liver
 - Liver infiltrates (e.g., amyloid or leukemia).
 - Cholangiolar obstruction in hepatitis (e.g., infectious, toxic).
 - Hepatic congestion due to heart disease.
 - Increased synthesis of ALP in liver.
 - Diabetes mellitus - 44% of diabetic patients have 40% increase of ALP.
 - **Liver diseases with increased ALP.**
 - 2× increase: acute hepatitis (viral, toxic, alcoholic), acute fatty liver, cirrhosis.
 - 5× increase: infectious mononucleosis, postnecrotic cirrhosis.
 - 10× increase: carcinoma of head of pancreas, choledocholithiasis, drug chole-static hepatitis.
 - 15-20× increase: primary biliary cirrhosis, primary or metastatic carcinoma.
 - Chronic therapeutic use of anticonvulsant drugs (e.g., phenobarbital, phenytoin)
 - Hodgkin's disease.
 - Preeclampsia, eclampsia, threatened abortion) but difficult to interpret without serial determinations. Lower in diabetic than in nondiabetic pregnancy.
 - Hyperthyroidism
 - Primary hypophosphatemia (often increased)
- **Normal In**
 - In acute icteric viral hepatitis, increase is <2× normal in 90% of cases, but when ALP is high and serum bilirubin is normal, infectious mononucleosis should be ruled out as cause of hepatitis.
- **Decreased In**
 - Excess vitamin D ingestion

550

- Radio & Lab. | Page 27
- Hypothyroidism, cretinism
 - Pernicious anemia (PA) in one-third of patients
 - Celiac disease
 - Malnutrition
 - Scurvy
 - Zinc deficiency
 - Magnesium deficiency
 - Postmenopausal women with osteoporosis taking estrogen replacement therapy
 - Therapeutic agents (e.g., corticosteroids, trifluoperazine, antiemetic agents, some hyperalimentation)
 - Cardiac surgery with cardiopulmonary bypass pump

7. ERYTHROCYTE SEDIMENTATION RATE (ESR)

- **Use**
 - **Indicates** presence and intensity of an inflammatory process; never diagnostic of a specific disease. Changes are more significant than a single abnormal occurrence.
 - Detect occult disease (e.g., screening program), but a normal ESR does not exclude malignancy or other serious disease.
 - **ESR is said to be useful to** differentiate iron deficiency anemia (ESR normal) from anemia of acute or chronic disease alone or combined with iron deficiency, in which ESR is almost always increased.
 - **Extreme elevation of ESR** is found particularly in association with malignancy (most frequently malignant lymphoma, carcinomas of colon and breast), hematologic diseases (most frequently myeloma), collagen diseases (e.g., RA, SLE), renal diseases (especially with azotemia), drug fever, and other conditions (e.g., cirrhosis). In patients with cancer, ESR >100 mm/hr indicates metastases. Other causes of ESR >100 mm/hr are severe infections (osteomyelitis, SBE), giant cell arteritis, polymyalgia rheumatica, renal diseases.
- **Interferences That Increase ESR**
 - Macrocytosis
 - Hypercholesterolemia
 - Increased fibrinogen, gamma or beta globulins

551

- Technical factors (e.g., tilted ESR tube, high room temperature)
- Drugs (e.g., dextran, methyldopa, methysergide, penicillamine, theophylline, trifluoperidol, vitamin A)
- **Interferences that decrease ESR**
 - Polycythemia, vera or secondary
 - Abnormal RBCs, especially sickle cells; hereditary spherocytosis; acanthocytosis
 - Microcytosis (e.g., HbC disease)
 - Hypofibrinogenemia (e.g., DIC, massive hepatic necrosis)
 - High WBC count
 - Technical factors (e.g., short ESR tube, low room temperature, delay in test performance >2 hrs, clotted blood sample, excess anticoagulation)
 - Drugs (e.g., quinine [therapeutic], salicylates [therapeutic], drugs that cause a high glucose level, high doses of adrenal steroids)
- **Increased in**
 - Chronic inflammatory diseases, especially collagen and vascular diseases
 - Postoperative (may be increased for up to 1 mo), postpartum
- **Decreased in**
 - Congestive heart failure
 - Cachexia
 - High doses of adrenal steroids
- **Factors that do not affect ESR**
 - Body temperature
 - Recent meal
 - Aspirin
 - Non-steroidal anti-inflammatory drugs

552

8. ABG

- Normal arterial blood gas is 7.35-7.45
- The key principle is that primary changes in HCO_3^- are metabolic and in CO_2 are respiratory.
- **A simple method**
 - **Look at the PH:** is there an acidosis or alkalosis?
 - $\text{PH} < 7.35$ = acidosis.
 - $\text{PH} > 7.45$ = alkalosis.
 - **Is the CO_2 abnormal?** (N: 35-45 mmHg).
 - CO_2 is an **acidic gas** ↑ed with acidosis, ↓ed with alkalosis
 - If so, the problem is respiratory.
 - If CO_2 is normal with acidosis or ↓ed → the change is compensatory.
 - **Is the HCO_3^- abnormal?** (N: 22-28 mmol/L)
 - HCO_3^- is alkaline, ↑ed with alkalosis, ↓ed with acidosis.
 - If so, the problem is metabolic.
- **An example:**
 - Your patient's ABG shows:
 - $\text{PH} = 7.05$
 - $\text{CO}_2 = 15 \text{ mmHg}$
 - $\text{HCO}_3^- = 8.0 \text{ mmol/L}$
- **Answer:**
 - It is acidosis because PH is < 7.35
 - CO_2 is ↓ed so, it is compensatory
 - HCO_3^- is low (with acidosis) so, it is metabolic problem.
 - So, it is a metabolic acidosis.
- **The anion gap:**
 - The blood +vely charged ions (**Cations**) must be balanced by -vely charged ions (**Anions**) → Anions = Cations to maintain electro-neutrality.
 - But when the main Cations ($\text{Na} + \text{K}$) are compared with main anions ($\text{Cl} + \text{HCO}_3^-$), there appears to be a shortage of anions or (Anion gap)

553

- **This gap is made of unmeasured anions** such as phosphate, sulphate, and -vely charged proteins (these are difficult to measure).
- **Anion gap** = $(\text{Na} + \text{K}) - (\text{Cl} + \text{HCO}_3^-) = \text{N: } 10-18 \text{ mmol/L}$
- A raised anion gap ($> 18 \text{ mmol/L}$) therefore indicates the presence of ↑ed unmeasured anions e.g. lactate, salicylate.
- **Causes of metabolic acidosis and increased anion gap:**
 - Lactic acid (shock, infection, hypoxia)
 - Urate (renal failure)
 - Ketones (diabetes mellitus, alcohol)
 - Drugs/toxins (salicylates, biguanides, ethylene glycol, methanol).
- **Causes of metabolic acidosis and normal anion gap:**
 - Renal tubular acidosis
 - Diarrhea
 - Drugs (acetazolamide)
 - Addison's disease
 - Pancreatic fistulae
 - Ammonium chloride ingestion.
- **Metabolic alkalosis: $\text{PH} \uparrow$ & $\text{HCO}_3^- \uparrow$**
 - Vomiting
 - K^+ depletion (diuretics)
 - Burns
 - Ingestion of base.
- **Respiratory acidosis: $\text{PH} \downarrow$ & $\text{CO}_2 \uparrow$**
 - **Type 2 respiratory failure** due to any lung, neuromuscular or physical cause.
 - Most commonly **COPD**. Look at the PaO_2 . It will probably be low. Is O_2 therapy required? Use O_2 if COPD is the underlying cause, as too much oxygen may matters worse.

554

- Beware exhaustion in asthma, pneumonia & pulmonary oedema which can present with this picture when close to respiratory arrest. These patients require ITU review for ventilator support.
- **Respiratory alkalosis: $\text{PH} \uparrow$ & $\text{CO}_2 \downarrow$**
 - A result of hyperventilation.
- **CNS causes:**
 - Stroke, sub-arachnoid hemorrhage, meningitis.
- **Other causes:**
 - Anxiety, altitude, fever, pregnancy, drugs, e.g. salicylates.
- **Test yourself**
 - **History**
 - A 35 years old woman with **type 1 DM** is brought to ER by ambulance after being found in her house **severely unwell**.
 - Following a discussion with her partner, she has **not been eating for the past few days** due to a vomiting illness and, as a precaution, has **also been omitting her insulin**.
 - Examination she appears **drowsy & peripherally shut down**, with **very dry mucus membranes**.
 - Her breath smells of acetone and her respirations are **deep and sighing**.
 - Pulse = 130 bpm
 - BP = 100/60 mmHg
 - RR = 26 breaths/min.
 - Blood glucose = $> 450 \text{ mg/dl}$
 - Free chest & abdomen.

555

14

• ABG

		Normal
H ⁺	88.9 nmol/L	(35-45)
PH	7.05	(7.35-7.45)
PCO ₂	11 mmHg	(35-45)
PO ₂	187 mmHg	(>80)
HCO ₃	6.0 mmol/L	(24-30)
SPO ₂	99.8 %	(>98%)
Lactate	1	(0.4-1.5)
BE	-25.2 mmol/L	(-2 - +2)
K	4.6 mmol/L	(3.5-5)
Na	141	(135-145)
Cl	96	(95-105)
iCa	1.25	(1-1.25)
Hb	12 g/dl	(13-18)
Glucose	630 mg/dl	(70-110)

► Questions:

- Describe her acid-base status?
- Calculate the anion gap?
- What is the most likely diagnosis?

► Answers:

- Severe metabolic acidosis with partial compensation.
- Anion gap = $(141 + 4.6) - (96 + 6) = 43.6$ (N: 10-18)
- DKA
 - Severe insulin deficiency leads to hyperglycemia & ↑ed metabolism of fats.

14

- The breakdown of fats produce **ketone bodies** (small organic acids which provide an **alternative source of energy** but can accumulate leading to a **profound metabolic acidosis**.
- It is the ketone bodies that can account for a **raised anion gap**.
- Beware of **severe dehydration**.

References

1. OHCM: p 684
2. ABG made easy p 86,87,123

تم فصل الأمثلة والتحليل والله تعالى الفضل والمنة